THE PHILADELPHIA NEUROLOGICAL SOCIETY.

October 27, 1903.

The President, Dr. H. A. Hare, in the Chair.

Hereditary Chorea.—Dr. W. W. Hawke presented a paper on this subject. The patient, a white male, aged thirty-eight years, was admitted to the Insane Department of the Philadelphia Hospital, April, 1903. His father had been an inmate of the same institution a few years previously, suffering from traumatic organic mania following the kick of a horse; shortly after the accident chorea developing. A paternal uncle died in the same institution at the age of forty-seven; this patient had dementia, and chorea developed about the time his mental symptoms commenced. Another paternal uncle had chorea and died insane. The patient's mother is living and well. He has had three children. One died at the age of nine years from "fits" after a short illness. The others are living and well. His symptoms began two years ago with twitching of the fingers and a tendency to become abusive. He acted foolishly. At present he is quiet but not melancholiac. He has general marked choreiform movements in the arms and legs and also in the face. The knee-jerks are increased. He has a spurious ankle-clonus. The pupils are slightly dilated. Eyegrounds are negative. The heart is negative. There is no history of rheumatism.

A Case of Primary Neuritic Atrophy.—This patient was exhibited by Dr. F. X. Dercum. He was a male, single, twenty-eight years of age, an insurance collector by occupation. The father is living and well. The mother suffered from epilepsy for a number of years. A paternal uncle had paresis. From the age of eighteen to twenty-nine years the patient used alcohol excessively. A venereal history is denied.

He dates the onset of the present trouble eight years ago. He had a chill followed by fever, and was sick about a week, but remained at home for about six weeks. This attack was followed by weakness in the limbs, requiring him to hold on to something when walking. Later he improved and was able to work for about two months. He was then obliged to give up his occupation on account of the difficulty in walking. He also complained of some shooting pains in the legs. He never had trouble with the sphincters. He has wasting of the legs below the knees and possibly some of the thighs, also wasting of the thenar and hypothenar eminences of the right hand. He also has scoliosis. A marked degree of ataxia is most distinct in the legs, but is also present in the arms. The reflexes of the lower extremities are absent, and those of the arms impaired. He has no sensory loss except a slight retardation of sensation. He has slight facial inequality. He has a distinct nystagmus. He shows no mental derangement. The eye report is negative. The nerve trunks seems to be more readily felt than they are normally.

Dr. A. Gordon remarked that he had some time ago exhibited a similar case of primary neuritic atrophy. That case presented many points similar to the one shown tonight. The symptoms began with neuralgic pain. The muscular atrophy in the peroneal group was very marked. The knee-jerks were lost. There was Argyll-Robertson pupil and also bladder involvement.

In the case shown tonight the onset at the age of fifteen years, the lost knee-jerks, the marked ataxia, the speech defect and the marked nystagmus would suggest the possibility of Friedreich's ataxia. The onset, the atrophy of the limbs and the peroneal atrophy favor the idea of primary neuritic atrophy.

The occurrence of nystagmus and of speech defect indicate that the primary neuritic atrophy as described in the text-books is rarely met with. The disease could more properly be named spinal-neuritic atrophy, because in association with the purely neuritic symptoms we also have spinal symptoms. Whether the initial lesion is in the nerves or in the spinal cord cannot be decided even with the pathological data.

Dr. William G. Spiller remarked that this was certainly not a typical case of the progressive, interstitial, hypertrophic neuritis. The symptoms in that disease are so striking that it is impossible to make a mistake in a typical case. He had examined the specimens from the first case of this disease reported by Dejerine and Sottas. He also had seen the brother of that patient and later had seen another case of the same disease in the service of Professor Dejerine.

Dr. F. S. Pearce said that this patient had reported at the Orthopedic Hospital some ten years ago. He was then considered a case of ataxia. The speaker thought that it was a case of Friedreich's disease. The patient denied having had syphilis. He said that he had had a severe attack of malaria, and that shortly after this the symptoms developed. This would suggest the possibility of malarial poisoning being the exciting cause of the disease.

Dr. F. X. Dercum remarked that the history was not that of Friedreich's disease. The disease was not hereditary. The man gave a history of a distinct febrile attack followed by neuritic pains and atrophy. Dr. Dercum was inclined to consider the case one of primary neuritic atrophy.

Some Graphic Observations on Ankle Clonus.—This paper was read by Dr. A. A. Eshner.

Dr. Charles K. Mills raised the question as to whether or not the movement produced by voluntary effort, as by standing on the ball of the foot, was a true ankle clonus. He regarded it as a movement of the entire leg.

The President remarked that according to the charts the reinforcement of the so-called clonus seemed to be characterized by an increase in the speed rather than by an increase in the excursion. In a true reinforcement he thought that the excursion would be increased rather than the speed.

Dr. F. S. Pearce asked whether or not in the experience of the author the rapidity of the clonus was in proportion to the spasticity of the patient.

Dr. A. A. Eshner said that in the paper he had expressed a doubt whether the movement obtained in a healthy person on supporting the weight of the body on the ball of the foot was a true clonus. The fact that under such circumstances, the movement is in the leg rather than in the foot is of little relevancy. It is the flexion of the leg on the foot; the leg moves on the foot because the former is free, the latter fixed. In the presence of ankle clonus the movement is the same, only the foot, being free, is flexed. In each instance the movement is the same, though the procedure is reversed.

With regard to reinforcement, Dr. Eshner had made a large number of observations before he could convince himself that any change could be induced in the frequency or the range of movement by extraneous causes. Finally he did observe that there seemed to be a change on attempts at reinforcement. To the hand, this seemed to be an increase in intensity, although in the tracings it appears as an increase in frequency. The failure to show in the tracing an increase in amplitude might be due to mechanical reasons, the movement of the patient's foot being restrained by the hand of the examiner or otherwise in the process of obtaining the clonus. It is possible that the greater the vigor of the clonus, the greater the pressure of the hand, so that the difference would not appear in the tracing as a change in amplitude.

Dr. Eshner had not studied the cases from the point of view suggested by the question of Dr. Pearce. He was not able to say whether the amplitude or the frequency bore any relation to the spasticity of the case.

A Case Resembling One of Progressive Spinal Muscular Atrophy.—Dr. C. S. Potts exhibited a patient, a man aged forty-nine years, a stone-cutter by occupation, who had never used liquor. A year ago, he noticed weakness in the left leg, manifested by a tendency to stub the toe and difficulty in getting the toe off the ground. Six months later, he noticed weakness in the right arm.

Examination shows the movements of the right arm to be impaired. The muscles most affected are the deltoid and biceps and possibly the triceps. In these muscles there is atrophy and a marked fibrillary tremor. Fibrillary tremors are also present in the corresponding muscles on the left side, but they are not so marked. At times, there is a violent painless cramp in the right biceps. In the left, the impairment of movement is most marked in the tibialis anticus and peroneal groups. There is no atrophy in the muscles of the leg, the circumference of the left leg in fact being greater than that of the right. The tendon jerks in the right leg are diminished. In the arms, the biceps jerk is present, but the other tendon jerks can not be elicited. There are no sensory symptoms. The electrical reactions are about normal. There is no weakness of the left arm or right leg.

The symptoms suggest both progressive spinal muscular atrophy and pseudo-muscular hypertrophy, the latter being suggested by the fact of the muscles of the affected leg being apparently better developed than those of the other. A dystrophy beginning at the age of forty-eight years would, however, be most unusual. Also the fibrillary tremors seen in the atrophied muscles of the arm are much more violent than are usually seen in cases of progressive spinal muscular atrophy. The symptom-group is certainly most unusual. No positive diagnosis has been made.

Dr. Charles K. Mills said that the case in some of its features seemed to be unique. Being unique, it is difficult to fix a pathology. The idea of myopathy suggests itself, but there are points against this view. It certainly is not a myelopathy of the ordinary type. He had never seen a case in which this peculiar form of cramp and local spasm occurred. It might possibly be a form of chronic poliomyelitis with irregular location of the lesions, in the lumbar cord on one side, and in the upper cord on the other, but even to this view there might be objection. It might possibly be an unusual form of gliosis of the cord affecting the ventral region.

Dr. F. X. Dercum suggested that pieces of the muscles be excised for microscopical examination.

Tabetic Symptoms in a Girl of Seventeen, Presenting from Infancy a Brachial Monoplegia, Probably of Poliomyelitic Origin.—This paper was read by Dr. A. Gordon. The patient, a girl, now seventeen years of age, at the age of eighteen months had an attack of fever and vomiting, followed by paralysis of the right arm. She remained well until three years ago, when the present trouble began with weakness in the left leg, with numbness in the thigh and leg, and especially in the sole of the foot. There was a continuous ache in the left leg and some lancinating pain. A year ago, the right leg became affected. There was also girdle-pain, some disturbance of micturition and weakness of vision.

The right arm is wasted and paralyzed. The right lower extremity is apparently normal in size, the left thigh is reduced. There is diminished response to the faradic and galvanic currents. Station is normal. The knee-jerk is lost on the left, and very much diminished on the right side. Objective sensation is diminished on the left side. Subjective sensation is more marked. There is marked weakness and numbness of the left leg, a sensation of tightness in the left lumbar region, imperative micturition and unequal pupils. The speaker expressed the opinion that this was probably a case of juvenile tabes.

Dr. Charles K. Mills considered the view well taken that this was a case of juvenile tabes with hereditary syphilis as its origin. He did not see any connection between the poliomyelitis and the juvenile tabes. They seemed to be a chance concurrence in the same patient. It may be that the etiology of the two conditions was the same.

Dr. A. Gordon said that he could not state positively that the poliomyelitis and the tabes bore the relation to each other of cause and effect. He reported the case as a curious example of these two diseases occurring in the same patient. Progressive muscular atrophy and amyotrophic lateral sclerosis have been reported as following acute poliomyelitis of infancy, but tabes has not yet been observed following this disease.

A Case of Solitary Tubercle of the Pons, with Paralysis of Associated Lateral Movement of the Eyeballs Toward the Left.—This case was reported by Dr. C. S. Potts and Dr. William G. Spiller. The patient was a man aged fifty-five years. The symptoms were: Weakness of the right arm and leg; and of the left side of the face, excepting the orbicularis palpebrarum; weakness of the muscles of mastication on the left side, with fibrillary tremors of these muscles; weakness of the left external rectus, with the loss of the power of associated movement of the eyes to the left, but with preservation of the power of convergence; diminution of the sensation of touch, pain, heat, and cold in the right arm, leg, side of the trunk, neck, occipital region, and ear; astereognosis and loss of the sense of position on the right side; diminution of the sensation of touch, pain, heat and cold on the left side of the face and head; slight loss of the sensation of heat and cold on the right side of the face; anesthesia of the conjunctiva, mucous membrane of the nose, mouth and tongue on the left side; deafness in the left ear and loss of the sense of taste in the left anterior half of the tongue; and some ataxia of both legs, most marked in the right. A tuberculoma involving the left side of the pons, but not invading either sixth nucleus, was found at the necropsy.

Three Cases of Injury of the Lower Spinal Cord, or Cauda Equina.—Dr. T. H. Weisenburg reported these cases. The first was that of a man who was struck by an engine and had paralysis of both lower limbs. At the present time, twenty months after the injury, the patient shows wasting of the buttocks and of both lower extremities, more marked on the right side. The weakness is greater on the right side, but the man can walk. No movements are possible in either ankle joint, and there is bilateral foot-drop and a typical steppage gait. Sensation is not normal. The patellar jerks are much exaggerated, ankle clonus is persistent, and Babinski's sign is obtained on each side. The bladder and bowel functions are normal. The lesion is placed in the anterior horns and between the 4th lumbar and 3d sacral segments, from where the muscles of the peroneal region are innervated. This region of the spinal cord has been named by Minor the epiconus. No similar case has been recorded by English or American writers.

In the second case a bullet entered at the 5th lumbar vertebra and its course was downward. There is a resulting paresis of the flexors of the right foot, and hypesthesia for touch and pain in right lower limb, corresponding to the distribution of the 2d, 3d, and 4th sacral segments. Both patellar jerks are increased. The Achilles jerks and Babinski sign are absent. Four similar cases have been found in the literature, where a lesion below the reflex arc caused an increase of the patellar tendon reflexes, but no special attention is placed upon this phenomenon by any writer. It is probable that some of the exito-motor fibers for the patellar reflexes exert their influence upon the segments above that in which they arise.

The third case was that of a man who fell one hundred and twenty feet, sustaining a fracture and dislocation of the 12th thoracic and 2d lumbar vertebræ. The right lower limb was paralyzed, and all the reflexes were absent, i.e., patellar and Achilles jerks and Babinski's sign. At the present time, three months after the injury, there is much restoration of motor function, the tendon reflexes are still absent. There is hypesthesia for touch and pain over the whole right lower limb, while on the left side there is hypesthesia for touch and pain only over the scrotum and penis. This form of the Brown-Séquard syndrome is possible only where the lesion is unilateral and is low in the spinal cord. Here the sensory fibers from the lower limbs have not as yet decussated, and therefore sensation and motion are disturbed in the same limb, while the sensory fibers supplying the scrotum, penis and perineum have decussated. Only one similar case has been found in the literature, that of Wernicke, afterward more fully detailed by Mann.

Dr. W. G. Spiller, referring to the first case, said that he had been much interested in the case for one or two years, because it is so exceptional to have steppage gait from spinal cord disease. This usually is a result of neuritis, and, as a rule, alcoholic neuritis. This man had a history of injury of the spinal cord. We must assume that only the gray matter of the fifth lumbar and first and second sacral segments was affected because bladder and rectal control was still present. The white matter had escaped. The knee-jerks were present and exaggerated, so that the lesion could not extend above the fourth lumbar segment. He did not know of a similar case in American or English literature. The only cases reported have been in Germany, and the area of the cord affected has been described as the epiconus. In the case reported by Dr. Weisenburg sensation was disturbed only over the dorsum of the feet and the outer side of the legs.

With regard to the second case, Dr. Spiller said that he had been waiting for some time to see an instance of exaggerated patellar reflexes from a lesion below the reflex arc. In this case there seemed to be little doubt as to the site of the lesion, as the bullet entered at the fifth lumbar vertebra and went downward as shown by the X-ray; yet this man had exaggerated patellar reflexes.

A Note on Section of the Fifth Nerve for the Relief of the Pain in Some Cases of Brain Tumor.—A note on this subject was presented by Dr. Charles K. Mills and Dr. C. H. Frazier. The authors suggested that in some cases of brain tumor, when the tumor could not be removed, the atrocious pain might sometimes be relieved by section of the fifth nerve. The main general symptoms of brain tumor are severe head pain, vertigo and vomiting. These symptoms are probably referable in most cases to irritation of the fifth nerve in the dura. This suggestion had been followed out in one case by Dr. Frazier with apparent success, as the pain has entirely disappeared.